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A Contribution to the Study of Atrophic Rhinitis.

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Bosworth, in his *Diseases of the Nose and Throat* (vol. i, pp. 162, 163), advances the following theory with regard to atrophic rhinitis:

Commencing in a desquamative inflammation of the nasal mucous membrane, there is set up a purulent rhinitis which constitutes the early stage of atrophic rhinitis. Later in the progress of the disease the discharge lodging upon the turbinated bodies forms a closely adhering film. In drying, this film contracts, with the result that the circulation of the blood in the large venous sinuses of the turbinated bodies is hampered, the nutrient supply of the membrane is shut off, as is that of the bones themselves, and all the structures of the bodies waste until they become scarcely recognizable as ridges upon the outer wall. The potent causative factor in this scheme is manifestly the contraction of the film. Without questioning its contractile power or its ability to strangle structures which are intended to contract (though it is difficult to conceive of such a possibility, unless extreme intrinsic contraction has already taken place),

it is plain that on this hypothesis, in the presence of dry air suspending dust, these films should be thicker, harder, and more destructive, and that every case of atrophic rhinitis should enforce the truth of this proposition.

In Colorado and throughout the dry belt of the United States the air is abnormally dry and at all times holds in suspension an immense amount of dust; therefore, under the best conditions for its demonstration, every case of atrophic rhinitis occurring in this territory ought to fully exemplify this teaching. Opportunities for observation are not wanting, but facts to prove this theory are. This disease is exceedingly common in those who have spent much time in Colorado, and while dry air and alkali dust have been the factors in production, it has not been through drying of discharge that they have exerted their influence, nor have they had or needed any gratuitous assistance from a previous purulent disease. Restricted to this single theory of causation we would not be able to explain one in a hundred cases of this disease existing here. Something else is indicated; something besides a bare proposition in mechanics is needed for the ninety-nine. We have in the study of atrophic rhinitis to do with a complex problem the equations of which may differ in different localities, but to which in Colorado dry air and alkali dust will usually give the key. These irritants, dry air and alkali dust, act as might be expected of them—the dry air intrinsically in demanding on the part of the secretory apparatus a larger amount of fluid, and the alkali dust by inciting to the parts a larger quantity of blood. These agencies, unceasing in their action, make their influence felt at the entrance of the respiratory tract, and as long as the turbinates sensibly respond to this excitation they bear its brunt. There is a time, however, when the turbinates do fail to meet these demands and when they not only cease to grow, but begin to waste. Of

course, if this process could be demonstrated step by step, the relation of hypertrophic to atrophic rhinitis might be determined once for all, but in the very nature of things this is impossible. To be permitted to observe a case for years, or even months, without doing anything for its relief, or allowing the patient this privilege, is a concession granted only by martyrs to science, and, so far, such patients I have not been called upon to treat. In the absence, therefore, of ocular proof of the various stages occurring in a single case, we must content ourselves with the observation of numbers of cases and with seeking for some principle that underlies them all. As a result of a careful study of the disease as it occurs in Colorado, it is certain that the presence of dried discharge has nothing to do with its inception and very little indeed with its continuance; for, while wasted turbinates are very often observed in those who have spent a number of years in this State, an almost invariable absence of noticeable fœtor or an accumulation of scales is suggestive of a physiological rather than a mechanical process. The observation of these wasted structures free from the classic accompaniments of stench and dried discharge enforces the idea that a hypertrophic stage must have preceded the present, and that, as a result of continuous and excessive stimulation, this has passed into one of exhaustion and of waste. This inevitable impression gathers force as the result of treatment is closely watched, for only on this assumption can the rapid approach to a healthy condition and appearance be explained. Only on the theory that we have to do in these cases with a condition of extreme exhaustion, rather than with one which, cutting off the supply of blood, is essentially necrotic, can we account for the easy reaccumulation of substance and the ready return to a condition of comparative health. In this view of the subject, made up as it is of the blending of many cases, the narration of

the individual is a matter of slight importance, yet, as an illustration, it may even here have an appropriate place.

Miss C., the daughter of parents whose intelligent answers to inquiries entitle to respect the mother's assertion that, prior to an attack of diphtheria during the past winter, the patient never had any nasal disease, has well-marked atrophy of the turbinates, with hypertrophy of the faucial tonsils and general thickening of the pharyngeal walls. Subsequent to the illness above noted she had nasal discharge for a short time only.

Miss T., the daughter of a prominent physician of this State, and so under constant medical surveillance, has never had purulent rhinitis, though at present unmistakably suffering from the atrophic form.

Mr. G., recently an official of the D. & R. G. R. R., remembers a time when the nasal passages were closed, but presents for treatment a case of atrophic rhinitis, with supersensitive areas of the sæptum.

In the case of Dr. D. there is an interesting condition of turbinated hypertrophy on the right side associated with such marked atrophy on the left that the inferior turbinated is scarcely distinguishable from the general contour of the outer wall.

These cases, selected simply as types from a larger number belonging naturally to the one or the other class, offer forceful lessons in ætiology. All these patients have spent a number of years in Colorado, some of them their whole life, and in each of them, though the condition is so plainly written that "he who runs may read," yet in not a single one of them was there at the time of their first observation distressing scaling, nor was fœtor present in the slightest noticeable degree. This condition of physiologic atrophy (if such it may be termed) gives the strongest clew to the *raison d'être* of the pathologic form. But we are not wholly dependent upon theory for the usual course of atrophic rhinitis, for, by one of those rare strokes of fortune which more than once has done service to the cause of medicine,

Dr. Clinton Wagner is able to record a case of hypertrophic rhinitis which, during an interval of several months from under his observation, passed into the atrophic form. To the mass of special students scattered throughout the land no argument is required to establish either the credibility of this witness or the competence of his testimony, and yet one author at least, for some inscrutable reason, has called the report in question.

As the object of this paper, offered simply as an attempt at explanation of the conditions that confront its author in daily practice, is not controversial in any sense, it is not necessary to further weigh the positive assertion of one observer against the theoretic statement of another. In the light, however, of the above-mentioned experience, Dr. Wagner's observation supplies the one necessary link to bind the two conditions together. On this positive basis of observation it becomes easy to follow the course of disease through the hypertrophic to the atrophic form; without this there is absolutely no way to satisfactorily explain the existence of atrophic rhinitis. It need not be denied that purulent rhinitis may result in atrophy, but doubtless in these cases the structures involved are destroyed by the severity of the original process, as in the case of diphtheria just related, to which the drying of discharge is an incident without moment. It is certainly logical to assume that in this case the acute disease enlarged before it finally, by its severity, overwhelmed these bodies. Except in point of time, it is not likely that the course of disease differed in this case from that in any other. Certainly purulent rhinitis must have a beginning, and from the moment of its inception the turbinates are subject to *ceaseless* stimulation, which must result in increased activity, in hypertrophy, and, if this stimulation is of sufficient duration, in exhaustion and waste. In the continuous character of the stimulation is

the principle underlying every condition that results in atrophic rhinitis. It seems hardly necessary to point out that under these circumstances any increase in size must be in a double sense abnormal, and is in itself the strongest evidence of exhaustion.

Such hypertrophy, rapidly manifested, evinces the attempt of Nature to keep pace with the ever-acting stimulation, and its continuous increment of size is the measure of its failure. The limit must be soon reached and wasting the inevitable result.

To clearly realize between the effect of continuous and intermittent stimulation, we have only to examine the pharynx and the region of the pharyngeal tonsil. In these cases of physiologic atrophy, as I have taken the liberty to call them, we find hypertrophy of the pharyngeal tonsil, enlargement of the follicles of the pharyngeal walls, and varicose pharyngeal veins. So constant is this association that to see the one is with certainty to predict the other. Nor is the connection between them difficult to trace. The pharynx, at first protected by the turbinated structures from the immediate causes of irritation, receives its impress at second hand. Thus its stimulation is slight or intermittent and its gradual development is determined; but, as the turbinated hypertrophy gives way to atrophy, its growth is slowly continued until finally, when the turbinated bodies have practically disappeared and the whole work devolves on it, and when for the first time it receives the whole shock of irritation, it is in a condition of perfected growth very different from hypertrophy the result of acute and ceaseless irritation. This hypertrophy, occasioning endless annoyance, may, without correction, persist for years.

Sir Morell Mackenzie and Lennox Browne have both credited dust with the causation of nasal and pharyngeal diseases. John Mackenzie, as quoted by Dr. Browne,

thinks that comparatively few cases of inflammation originate in this way; but I do not recognize in his failure to trace the connection between them the least objection to this view, for the city of his residence is noted for its many hills, its surface drainage, and its frequent rains, conditions that by the grace of God, despite the health authorities, insure clean streets and the minimum of dust, and his opportunities to note the effect of this agent are therefore necessarily limited. Were the field of his observation surrounded by many hundred miles of arid plain, he might find it necessary to change his opinion.

I am convinced that under these conditions, and traceable primarily to this cause, there exists a low form of inflammation of the turbinated bodies which eventually results in their atrophy; that the later stages of the disease give every evidence of having been preceded by hypertrophy; that the subjects of this disease present themselves for treatment of the throat, either entirely ignoring the nose or referring to it merely in an incidental way; and, finally, that this variety of atrophic rhinitis by its simpler process offers the logical explanation of the more complicated form.

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